blood changes and in part from cardiac failure), respiratory and cardiac failure; the prime cause, of course, being the excessive amount of body heat, the others depending partly upon it and partly upon each other, forming to a certain extent a vicious circle. The blood, probably, was first altered in composition (semi-baked), causing impaired nutritive capacity with a tendency to stagnation from its viscidity. The renal capillaries became blocked up by the altered blood, thus cutting off the excretion of urea and its allies, which must have been formed in enormous quantity over and above that of health, from the rapid metamorphosis of tissue which it is reasonable to expect was occurring under the influence of the high temperature. Venous stagnation and vitiated blood, together with the changes in the cerebral tissue incident to the high temperature, produced the coma with the impairment of activity in the respiratory centre, while the latter, with venous stagnation and cardiac weakness, caused interference with respiration. Respiratory interference again reacted upon both the cerebrum and the heart. So, also, the heart, organically affected by the high temperature and functionally by the changes in other organs, kept the vicious circle revolving.

The secretions of the skin were markedly absent, and the intestinal tract also was greatly disturbed, as evidenced by the frequent occurrence of profuse discharges.

But a few of the cases could be traced after their departure from the hospital. Of those who were reached, Cases IV., V., VII., XXII., and XXIII. have had no sequelæ, while Case VIII. has had impairment of vision and memory.

ALCOHOLIC PARALYSIS.

By C. W. Suckling, M.D. Lond., M.R.C.P.,
PHYSICIAN TO THE QUEEN'S HOSPITAL, BIRMINGHAM.

The earliest account of alcoholic paralysis is said to have been given by Dr. Jackson, of Boston, in 1822. Very little attention was given to Dr. Jackson's observations, which, however, were confirmed, in 1849, by Magnus Huss, of Stockholm. Dr. Wilks was one of the first British writers to draw attention to the frequency of paraplegia in inebriates, and he wrote, in the *Lancet*, on the subject fifteen years ago, and described it in his *Lectures on the Diseases of the Nervous System*, published in 1878. Since Dr. Wilks's papers alcoholic paralysis has been accurately described by numerous observers; and the lesion of the nervous system associated with this paralysis was proved by Lancereaux, in 1881, to be a multiple peripheral neuritis. Dreschfeld and Hadden, in this country, have confirmed the discovery of Lancereaux, and it is certain that the paralysis in chronic alcoholism is due to changes in the peripheral nerves. Déjérine, Charcot, Hun, Buzzard, Handfield Jones, and Broadbent, have all reported cases, and have each added to our knowledge of the disease. As the result of the labors of these observers alcoholic paralysis is now acknowledged to be a definite malady with well-defined symptoms and pathology.

Dreschfeld (*Brain*, vol. vii.) divides alcoholic paralysis into two types: alcoholic ataxia and alcoholic paralysis proper.

Alcoholic ataxia occurs chiefly in man, and is by no means so frequently met with as is alcoholic paralysis. The symptoms greatly resemble those of locomotor ataxia, there being marked incoördination in the lower extremities, lancinating pains, and loss of knee-jerk, without paralysis or atrophy. In all cases in which there is loss of the knee-jerk and ataxia, alcoholism must be excluded before the diagnosis of tabes is arrived at.

In alcoholic ataxia there is usually a history of alcoholic excess, but the absence of this cannot be relied upon, for it is often concealed or denied; men, however, not being so deceitful in this respect as women.

Morning sickness and dyspepsia, or a history of hæmatemesis, would be strong evidence in favor of the alcoholic origin of the affection.

Mental symptoms, such as loss of memory and delirium, would be suggestive; but loss of memory and other mental symptoms are common in tabes.

In alcoholic ataxia we do not find myosis, or the Argyll-Robertson pupil, or joint affections, or visceral crises, but the knee-jerk is abolished.

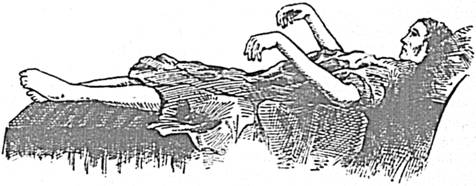
In alcoholic ataxia there is generally some tenderness in the calf-muscles, which, when present, points strongly to the alcoholic nature of the affection. Moreover, any qualitative electrical changes would be decisive against tabes.

The symptoms in this form of alcoholic paralysis are due to a multiple neuritis, especially of the sensory nerves, and it differs from the second type in the absence, or, at any rate, in the extreme mildness of hyperæsthesia, hyperalgesia, paralysis, and atrophy.

The second type of alcoholic paralysis, *alcoholic paralysis proper*, is by far the most common, and it is met with in women far oftener than in men. The disease usually commences and progresses gradually, being subacute in its intensity, but the onset is occasionally acute, and attended with fever. Sensory disturbances are well marked, there being hyperæsthesia of the feet and legs, which, in severe cases, may be followed by anæsthesia and analgesia. The patient usually complains bitterly of "cramps" in the calf muscles and of lancinating pains. These sensory disorders are usually most marked in the lower extremities, but frequently affect the upper limbs also. The calf-muscles are exquisitely tender when grasped, and this symptom is highly characteristic of the disease. Tenderness is, also, usually elicited by pressure applied along the course of the nerves of the lower extremities.

A remarkable perversion of the sense of temperature has been observed in the subjects of alcoholic paralysis, all objects in contact with the skin appearing cold; I have not observed this in the cases I have met with. The patient complains of weakness, or may be of absolute paralysis in the feet and legs, sometimes she is able to get about with a shuffling gait, being unable to pick her toes up properly, and, therefore, constantly tripping; in more advanced cases the patient lies in bed helpless, being unable to move her feet, but having still some slight power in the hands. The extensor muscles of the legs suffer earliest and to the greatest extent, the feet being drooped and flaccid, all power of dorsal flexion of the feet being usually lost. The knee-jerk is almost invariably, and the plantar reflex frequently, abolished.

The arms are affected later than the legs and to a much less degree; the extensor muscles here also suffering most, dropped wrist (see illustration) being occasionally, though by no means always, present.



Alcoholic paralysis.

The paralysis, though usually affecting only the extremities, and always commencing and being most severe in these parts, may, however, be general; and even when extensor paralysis alone is evident, careful examination shows that there is general muscular enfeeblement.

The cranial nerves are sometimes affected; in one of my cases the presence of squint together with vomiting led to the diagnosis of cerebral tumor.

Buzzard has met with paralysis of the soft palate, of the face, and also of the external rectus oculi; and Hadden has noticed nystagmus in alcoholic paralysis.

In one of my cases ophthalmoplegia externa was present, together with double optic neuritis. The common causes of ophthalmoplegia externa are locomotor ataxia, syphilis, diphtheria, and exposure to cold.

Though paralysis of the ocular muscles has been observed in chronic alcoholism, I am not aware that the condition to which the term ophthalmoplegia externa is applied has been met with, and its occurrence would seem to show that it may be produced by lesion of the nerves as well as by lesion of the nerve nuclei, alcoholic paralysis having been proved to depend upon peripheral neuritis.

The following case of chronic alcoholism with ophthalmoplegia externa and double optic neuritis is at present under my care:

J. B., a man, aged fifty, was admitted into the Birmingham Workhouse Infirmary on January 25th. His relatives state that he has been drinking heavily for some years, and my friend, Mr. Newton, who attended him before his admission into the Workhouse, tells me that he has been drinking for years, and that the drooping of the eyelids came on about a month before admission; the patient having complained some weeks previously of pains and cramps in his legs.

The patient on admission was incoherent, constantly asking for drink, and unable to tell where he was, or to give any account of himself. He was unable to raise his eyelids, there being drooping of both lids, the left being less affected than the right.

There was slight external strabismus of the right eye. He was unable to rotate his eyeballs either upward or downward, but could move them readily from side to side. The pupils were small, but responded to light and accommodation. There was a slight degree of optic neuritis, the disk on each side being blurred and the vessels very tortuous. The knee-jerk was lost on both sides, the plantar reflex increased. There was no paralysis of the legs or arms, but the calf-muscles were exquisitely tender on being grasped, and pressure along the course of the posterior tibial nerves elicited great pain.

He could point his toes and there was no marked weakness of the extensors of the wrist or leg. The first metatarsal bone was fractured and he said this was due to a chair falling on his foot. The muscles of the leg responded normally to faradism and galvanism. His memory was much affected and he did not know where he was.

He had no knowledge of time or place. When asked if he had been out he always responded in the affirmative, declaring that he had been several miles and that he had had several glasses of whiskey—in fact, he talked of nothing but drink.

He took his medicine readily on being told it was whiskey, though he thought the taste of it was "very peculiar." In fact, he thoroughly illustrates the truth of the proverb, "In vino veritas," his speech betraying his previous habits. Since admission he has much improved, being now able to open his eyes, the lids only drooping slightly. There is still considerable restriction of the movements of the eyeballs, but this is daily diminishing. The patient has had no alcohol since admission.

The paralyzed parts are always flaccid and atrophied, the degree of atrophy varying with the intensity of the neuritis. Contracture may occur, but is rare. The bladder and the rectum are rarely implicated. The faradic irritability in mild cases may be present in a normal degree, but is usually diminished and in severe cases is entirely abolished. The galvanic irritability is increased and the contractions sluggish and prolonged, the reaction of degeneration being well marked in severe cases.

Vasomotor changes are usually present, the extremities being red and sometimes œdematous and occasionally presenting purpuric spots, but these changes and also trophic disturbances are always more marked in the lower than in the upper extremities. Bedsores do not occur except in the worst cases.

The skin of the hands and feet sometimes presents the glossy appearance first described by Paget, at other times it is harsh and dry. The nails are frequently affected, being curved, ridged, opaque, and brittle.

Mental symptoms are almost invariably present; the memory is impaired especially for recent events; the patient is emotional, suffers from insomnia, dreams, restlessness, delirium, and hebetude, together with digestive disturbances such as morning sickness and retching. Such symptoms occurring with ataxy or paralysis render the diagnosis of the nature of these two symptoms tolerably certain.

I have elsewhere corroborated an observation of Dr. Dreschfeld's, of a peculiar form of delirium present in the subjects of alcoholic paralysis. Dr. Dreschfeld noticed that the subjects of this disease, though unable to move, would say that they had been out walking, often an impossible distance. Since writing on this delusion I have had abundant evidence of the truth of his observation.

In one case I asked the patient, who was quite helpless, if she had been out, and she said that she had been in the garden that morning—the friends also told me that she was always saying she had been walking somewhere—this delusion alone decided me in my diagnosis, though I had at that time not been acquainted with her drinking habits. This peculiar delusion is met with in most cases of chronic alcoholism. I had a man recently under my care at the Birmingham Workhouse Infirmary dying of phthisis, who had severe multiple neuritis—both legs being completely paralyzed. He had been a heavy drinker and it is probable that both the lung mischief and the neuritis had been brought about by alcohol, though Pitres and Vaillard have proved that peripheral neuritis follows phthisis apart from alcoholism.

This man, when asked if he had been out, always answered in the affirmative, and would declare that he had just walked several miles, mentioning localities in the neighborhood which he had just visited.

Within the last few months I have been called to see three women suffering from well-marked alcoholic paralysis, and I have no doubt such cases are of common occurrence, and that they are often unrecognized.

In all of these cases the drinking habits of the patient were unknown to the medical man in attendance, and even when he had prohibited stimulants, the patient's friends had yielded to her importunity and were supplying her with "spirits" up to the very day that I attended. They presented the usual symptoms of the disease; one had suffered from diplopia. I learned from their medical attendants that one died, but that the other two recovered. One patient could walk ten weeks after I saw her, though her gait was shuffling, and the knee-jerk still absent. Another had rapidly improved on the withdrawal of all alcohol, and though unable to stand when I saw her, in a few months she could walk. She soon had a relapse, which her doctor discovered to be due to her again taking alcohol. He tells me that he read the "riot act," and that she was soon able to walk about again. The patient who died was in the typhoid state and evidently too far gone to save. I fancy the abrupt discontinuance of the alcohol in this case may have accelerated death.

When the diagnosis is arrived at, nay, even when the disease is suspected, the patient's friends and attendants should be separately questioned, and search should be made in the patient's room, and even under the mattress and pillows of her bed for bottles or other evidence of her propensity.

The diagnosis is usually an easy one. The patient complains of pains and loss of power in her legs, the feet are drooped and flaccid, and she cannot point her toes upward. The power of dorsal flexion of the feet is diminished or absent. The grasp is feeble, but there is often no marked paralysis in the upper extremities.

Great pain is elicited if the calf muscles are grasped, and tenderness is present along the course of the nerve trunks, the patient complaining bitterly of cramps in the calf muscles.

It will be found that the knee-jerk is absent, and this is a most important symptom, as tabes is rare in women. Mental disturbance is always more or less present, there being usually considerable loss of memory, and the peculiar delusions already mentioned. The presence of sickness or retching in the morning is highly suggestive of the disease.

It must be remembered that in many cases no history of alcoholic excess is obtainable unless the medical man, from being sure of his diagnosis, insists on its alcoholic nature, so that the diagnosis at first must be made from the physical and mental state of the patient, the diagnosis required being first that the case is one of multiple peripheral neuritis, and then that the cause of this neuritis is alcoholic excess.

In well-marked cases the diagnosis of peripheral neuritis is very easy, the sensory disturbances and electrical changes showing that the lesion is one of the peripheral nerves, but in mild cases sensory alterations may not occur at all, for in neuritis of a mixed nerve the motor fibres always suffer most; frequently, also, the nerves are affected beyond the points at which their sensory branches are given off, and thus motor symptoms alone are present though the nerve is a mixed one.

Moreover, there may be complete motor paralysis without any electrical changes; but the distribution of paralysis is always an anatomical one, the muscles paralyzed being in groups supplied by particular nerves, the anterior tibial being the nerves especially affected, and the paralysis is gradual in its onset and progressive. By these features we can distinguish even these mild cases of peripheral neuritis from lesions of the anterior cornua of gray matter of the cord, but there is no doubt that many cases which have been recorded under the name of "subacute general spinal paralysis," have really been cases of peripheral neuritis.

The medical man should ask himself, Is the case due to organic or functional causes? If organic, is it due to cerebral, spinal, or peripheral disease?

The absence of the knee-jerk alone is quite sufficient to prove that the paralysis is not hysterical—the knee-jerk is never absent in simple hysteria and its absence denotes a lesion somewhere in the reflex path which traverses the cord at the level of the second and third lumbar nerves.

In cerebral paralysis there are no electrical changes, sensation is usually unaffected, rigidity and not flaccidity is present with exaggeration and not abolition of the tendon reflexes, muscular wasting is not marked, nor are trophic changes usually present.

Should a squint be present in a case of alcoholic paralysis it is possible that the vomiting and delirium might lead to the diagnosis of cerebral tumor or other brain lesion as the cause of the paraplegia. But this mistake is a bad one and the patient would in all probability be sacrificed through it, for she would be allowed to go on taking stimulants until she died.

The following diseases of the spinal cord must be distinguished from peripheral neuritis.

1. Locomotor ataxia.
2. Poliomyelitis anterior acuta.
3. Myelitis.
4. Meningitis.
5. Subacute general spinal paralysis.
6. Acute ascending paralysis or Landry's disease.

It is highly probable that cases of neuritis due to alcohol or other cause have been diagnosed as cases of locomotor ataxia and their cure as that of this almost hopeless disease. Loss of the knee-jerk, pains, and ataxy are present in both diseases, but the pupil symptoms of tabes are not present in peripheral neuritis. Qualitative electrical changes would be decisive against tabes. Before the diagnosis of tabes is made peripheral neuritis must be carefully excluded.

In peripheral neuritis due to alcohol the case runs a far more rapid course than does a case of tabes, and paralysis is soon very evident, with drooping of the toes and tenderness of the calf muscles.

In lesions of the anterior cornua of the spinal cord, as in subacute general spinal paralysis, there are no sensory disturbances and the distribution of the paralysis is a physiological one, muscles functionally associated being paralyzed, while in peripheral neuritis the distribution of the paralysis is an anatomical one. In multiple neuritis the extensor muscles of the forearms are always the first muscles of the upper extremities to be affected and the trunk muscles escape, while in ascending paralysis, due to spinal cord disease, the trunk muscles become paralyzed after the legs, and when the paralysis spreads from the intercostal muscles to the upper extremities the intrinsic muscles of the hand (supplied by the first dorsal nerve) are always the first to be affected, the extensor muscles of the wrist and fingers (supplied by the seventh cervical nerve) being affected later as the lesion ascends the cord.

In spinal meningitis the symptoms are those peculiar to lesions of the peripheral nerves, the nerve roots being implicated, but severe pain in the back aggravated by movement with retraction of the head and muscular twitchings distinguish this disease from peripheral neuritis.

In Landry's disease there are no sensory disturbances and no electrical alterations.

In paraplegia due to myelitis bedsores, bladder and intestinal troubles are almost invariably present, while in peripheral neuritis these symptoms are of quite exceptional occurrence.

The prognosis in alcoholic paralysis is usually favorable, if the nature of the disease is detected early, and its cause removed; should the patient still continue to obtain alcohol, the case will end fatally, either from bedsores or from some associated diseases, phthisis, or cirrhosis of the liver, or by the production of the typhoid state. Gowers has observed death from rapid cardiac failure in two cases. Complete recovery may ensue after the disease has continued for a year.

It is only by the use of electricity that we can make an accurate prognosis of the duration of the paralysis in a given case. If the faradic response be lost, and the reaction of degeneration be present, then the paralysis is severe, and several months must elapse before recovery can ensue. The gravity is increased should the galvanic muscular irritability be greatly diminished, and should there be no irritability at all, then recovery cannot take place. Of course, these remarks apply only to the degree of neuritis present. The general condition of the patient must be taken into account in giving an opinion as to the patient's chance of recovery. The presence of jaundice, fever, delirium, phthisis, or cirrhosis of the liver renders the prognosis more grave. The time in which recovery may ensue must always be reckoned in months. Twelve months frequently pass before recovery is complete.

In all cases it must be remembered that no improvement can take place unless the patient be prevented from obtaining drink, and all such patients should be watched, and deception must be expected and looked for. Rest in bed is necessary, and after acute symptoms have subsided galvanization of the affected nerves and muscles is useful. If there is much pain, hot fomentations and anodyne liniments are very useful. Cocaine may be applied locally or injected hypodermatically. Opium must be given with great care. Rubbing is of great value, particular attention being given to the extensor muscles of the toes, and by this means contracture of the calf-muscles is prevented, and recovery hastened.

Internally, iodide of potassium, with strychnia and iron, has seemed to do good in my cases. I consider strychnia a most valuable remedy in chronic alcoholism. The application of blisters and counter-irritants along the course of the nerve-trunks is also useful, but, of course, interferes with the rubbing, which I believe to be the most useful of all modes of treatment in this complaint. It is difficult to estimate the value of drugs in this disease, for probably the mere withdrawal of alcohol is quite sufficient for the cure of most cases.

A CASE OF ALCOHOLIC PARALYSIS,

IN WHICH MYALGIC PAINS AND TENDERNESS WERE ABSENT, AND IN WHICH THERE WAS VERY LITTLE DISTURBANCE OF THE CUTANEOUS (TACTILE) SENSIBILITY; WITH A REPORT OF THE MICROSCOPICAL APPEARANCES OF THE NERVES IN A CASE OF PERIPHERAL NEURITIS, AND PERFORATING ULCER OF THE FOOT, ASSOCIATED WITH DIABETES MELLITUS.

By Byrom Bramwell, M.D., F.R.S.E., F.R.C.P. Edin.,

ASSISTANT PHYSICIAN TO THE EDINBURGH INFIRMARY; LECTURER ON THE PRINCIPLES AND PRACTICE OF MEDICINE IN THE EXTRA-ACADEMICAL SCHOOL OF MEDICINE, EDINBURGH.

Sensory disturbances, more especially myalgic pains, muscular tenderness, and hyperæsthesia of the skin, are striking features in the great majority of cases of alcoholic paralysis. The following case is peculiar and exceptional inasmuch as myalgic pains and muscular tenderness were absent throughout its whole course, and that there was very slight evidence of any affection of the cutaneous (tactile) sensibility until the period of convalescence, when marked hyperæsthesia of the skin of the lower extremities was developed. The case is also interesting because